using more recent data is unlikely to change that conclusion (see Appendix of Jerrett et al. 2009). Finally, national risk estimates are more applicable globally than city-specific estimates because they include larger and more diverse populations.

However, because the evidence for chronic ozone mortality is more limited than the large body of evidence demonstrating mortality associations with short-term ozone exposure, we present here estimates of the global burden of ozone on mortality using RR estimates from Bell et al. (2004), a large multicity study of short-term ozone mortality. We estimated mortalities daily using the difference between preindustrial and present-day 8-hr maximum ozone, and sum mortalities over the 1-year simulation. We used the reported relationship for cardiopulmonary mortality and daily average ozone [0.64% (95% posterior interval, 0.31–0.98%) for a 10-ppb increase], and corrected to 8-hr ozone using the reported ratio between daily 8-hr and 24-hr average ozone associations with nonaccidental mortality.

Using these methods, we estimated 362,000 (95% confidence interval, 173,000–551,000) annual global premature cardiopulmonary deaths attributable to ozone, approximately 50% of the 700,000 premature deaths we calculated in our original study (Anenberg et al. 2010). Since estimated deaths due to $PM_{2.5}$ (3.7 million) are an order of magnitude larger, using a short-term rather than long-term RR estimate for ozone has only a minor effect on the overall global burden of disease due to outdoor air pollution. As RRs for chronic ozone mortality are not as strongly supported as those for $PM_{2.5}$, we expect that estimates of mortality burden will improve as research on chronic ozone exposure and mortality continues globally.

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## Influence of Selenium and Mercury on Age-Related Cataracts in the Brazilian Amazon

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In their article, Lemire et al. (2010) provided important data on the frequency of age-related cataracts among adults in the Brazilian Amazon and found a correlation between age-related cataracts and whole-blood total mercury (Hg) concentrations and selenium (Se) levels in plasma and whole blood. However, in the "Discussion" of their paper, they stated that they "observed no adverse effects although Se concentrations were very high, reaching 1,500 μg/L for [blood]-Se and 913 μg/L for [plasma]-Se" (Lemire et al. 2010). However, they did not mention the potential and substantial adverse health effects associated with a high body Se burden. There are potentially adverse consequences to Se body burden, such as hair loss (alopecia), tooth decay, nail changes, peripheral paresthesias, weakness, skin lesions, and diabetes (Hira et al. 2004; Nuttall 2006; Shearer 1975; Stranges et al. 2010; Sutter et al. 2008; Yang et al. 1983). It would be valuable to know what Se-related adverse effects were observed by Lemire et al. (2010). Most studies, taken together, suggest a possible attenuation of Hg toxicity, probably as insoluble form of Hg selenide (Clarkson 2002; International Programme on Chemical Safety 1990).

Selenium may be able to delay the onset of toxic effects in animal models exposed to methylmercury in the diet (Clarkson 2002; Ganther et al. 1972). However, the Hg–Se interaction may not have an equivalent effect in some animals. There is evidence that coadministration of methylmercury and Se may lead to an important synergistic effect (Heinz and Hoffman 1998). In studies of persons who have been co-exposed to Hg and Se, toxic effects of Se should be taken into account to discover the potential synergistic effect between Se and Hg.

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## Influence of Selenium: Lemire et al. Respond

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We agree with the comments made by Minoia et al. in their letter. In this study population, we did evaluate the sentinel signs and symptoms of selenosis (Lemire M, Philibert A, Fillion M, Passos CJS, Guimarães JRD,

Barbosa F Jr, Mergler D, unpublished data), and we observed no association between any of the biomarkers of selenium (Se) status and signs and symptoms of selenosis (hair loss, broken nail walls, nail sloughing, skin lesions, garlic breath, gastrointestinal disorders, and motor and sensory deficits), despite high Se body burdens. Other results in this study population show a positive association between Se status and motor performance (Lemire M, Fillion M, Frenette B, Passos CJS, Guimarães JRD, Barbosa F Jr, Mergler D, unpublished data).

Guidelines on Se toxicity (International Programme on Chemical Safety 1986; U.S. Environmental Protection Agency 2002) are based mostly on reports of chronic selenosis in a Chinese population with excessive Se exposure, resulting from high Se in crops fertilized with coal ash highly rich in Se (Yang et al. 1983), and from combustion of Se-rich coal for domestic use (Liu et al. 2007). Drinking water likewise contained unusually high concentrations of inorganic Se (Yang et al. 1983). Thus, several factors, such as exposure to toxic vapors from coal combustion and/or inorganic Se from drinking water, may have contributed to toxic Se effects observed in China, primarily attributed to high organic Se in local crops. Recent studies reporting selenosis most often refer to excessive Se intake from nutritional supplements or inorganic Se through drinking water (Sutter et al. 2008; Vinceti et al. 2010).

Our study (Lemire et al. 2010) was conducted in 2006, before the first publication on an association between Se supplementation and the incidence of self-reported type 2 diabetes (Stranges et al. 2007). In the Amazonian population in our study, only 5 of the 448 participants reported diabetes. Further studies should address the possible associations between high Se status and hypertension and hypercholesterolemia (reviewed by Stranges et al. 2010).

Other evidence comes from Inuit, whose traditional diet of marine mammals is exceptionally rich in Se. The prevalence of diabetes in Nunavik, Québec, Canada, is low (3.5%). Ferland et al. (2009) observed no association between Se status and diabetes or plasma fasting glucose and insulin levels. Inverse association between blood Se and systolic blood pressure has also been reported (Valera et al. 2009). Hansen et al. (2004) pointed out that there are no recorded signs of selenosis in Greenland populations and that their high Se intake may be tolerated at higher levels.

In Northern and Amazonian populations exposed to mercury (Hg), high dietary Se may offset Hg-mediated oxidative stress and/or be required to maintain optimal selenoenzymes. There may be consequently less "excess" Se and little or no Se toxicity (Khan and Wang 2009).

Whether dietary Se is less toxic in Hg-exposed populations remains unanswered. However, even if there is increasing evidence that Se can offset some toxic effects of Hg, it may be inefficient against all Hg-mediated effects. Preventive actions should continue to focus on reducing Hg exposure rather than increasing Se status.

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## Erratum

The February 2011 article "Avoiding Health Pitfalls of Home Energy-Efficiency Retrofits" [Environ Health Perspect 119:A76–A79 (2011)] incorrectly listed 1,2,3-trichloropropane (TCP) as a flame retardant used in spray polyurethane foams. The correct compound should have been tris(1-chloro-2-propyl) phosphate (TCPP).

*EHP* regrets the error.